

ALCOHOL MARKETING, ADOLESCENT DRINKING AND PUBLICATION BIAS IN LONGITUDINAL STUDIES: A CRITICAL SURVEY USING META-ANALYSIS

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Abstract. This paper presents a meta-analysis of prospective cohort (longitudinal) studies of alcohol marketing and adolescent drinking, which accounts for publication bias. The paper provides a summary of 12 primary studies of the marketing–drinking relationship. Each primary study surveyed a sample of youth to determine baseline drinking status and marketing exposure, and re-surveyed the youth to determine subsequent drinking outcomes. Logistic analyses provide estimates of the odds ratio for effects of baseline marketing variables on adolescent drinking at follow-up. Using meta-regression analysis, two samples are examined in this paper: 23 effect-size estimates for drinking onset (initiation); and 40 estimates for other drinking behaviours (frequency, amount, bingeing). Marketing variables include ads in mass media, promotion portrayals, brand recognition and subjective evaluations by survey respondents. Publication bias is assessed using funnel plots that account for ‘missing’ studies, bivariate regressions and multivariate meta-regressions that account for primary study heterogeneity, heteroskedasticity, data dependencies, publication bias and truncated samples. The empirical results are consistent with publication bias, omitted variable bias in some studies, and lack of a genuine effect, especially for mass media. The paper also discusses ‘dissemination bias’ in the use of research results by primary investigators and health policy interest groups.

Keywords. Adolescents; Advertising; Alcohol; Meta-analysis; Publication bias

Advertising influences youth drinking...[but] there is a possibility that publication bias may have affected the studies identified for inclusion. (Anderson *et al.*, 2009, pp. 13–14)

This systematic review... shows some evidence for an association between prior alcohol advertising and marketing exposure and subsequent alcohol drinking behaviour in young people...[but] we cannot rule out the possibility of publication bias. (Smith and Foxcroft, 2009, pp. 12–14)

Publication bias... is a serious problem in the interpretation of scientific research. (Begg and Berlin, 1988, p. 419)

1. Introduction

Concerns about the deleterious effects of alcohol consumption, especially by youth, exist in many developed countries (World Health Organization, 1999; Babor *et al.*, 2003). Public health policy in this area is often guided by empirical analyses conducted by several different scientific disciplines. Economic studies usually call for tax or price increases as a policy measure (National Research Council, 2004), but some research studies also advocate greater regulation of advertising (Saffer and Dave, 2006; Gordon *et al.*, 2009). Because definitive research results are difficult to obtain, there is considerable debate about the effects of alcohol advertising and marketing on youth drinking. For example, a review by the National Institute on Alcohol Abuse and Alcoholism (2000, p. 422) concluded that 'when all of the studies are considered, the results of research on the effects of alcohol advertising are mixed and not conclusive'. Another review by the National Research Council (2004, p. 134) found that 'a causal link between alcohol advertising and underage alcohol use has not been clearly established'. However, two recent reviews reach a different conclusion. These surveys examine prospective cohort (longitudinal) studies of marketing and adolescent drinking. Both surveys conclude that advertising plays a role, albeit modest, for the onset of alcohol use by adolescents and the frequency or amount of drinking (Anderson *et al.*, 2009, p. 1; Smith and Foxcroft, 2009, p. 51). Longitudinal studies examine self-reported alcohol consumption by youth, including effects of exposure to commercial messages in the mass media and other marketing methods (branded merchandise, movie portrayals, music videos etc.). The studies first interview a sample of adolescents in order to establish a baseline for marketing exposure and current drinking prevalence, if any. Second, the adolescents are re-interviewed – usually within 2 years – to determine drinking onset, frequency and other behaviours such as binge drinking. Third, the baseline data on exposure are used to estimate regression models of subsequent alcohol behaviours. Covariates in multivariate regressions include demographics, social influences to drink, personality traits and baseline marketing exposure.¹

Anderson *et al.* (2009) and Smith and Foxcroft (2009) are examples of systematic reviews, which are literature surveys focused on a single question that attempt to identify, appraise and synthesize all 'high-quality' evidence relevant to the question. The reviews are selective with regard to topic and research studies, rather than comprehensive surveys of broader subject matter. It is widely believed that systematic reviews minimize bias on the part of reviewers and impart reliability to research results by seeking to identify valid empirical studies. In the public health area, systematic reviews often use empirical studies from randomized controlled trials or those that employ longitudinal data and methods. It is common to argue that longitudinal studies identify causal relationships, and this identification is not possible with cross-sectional data and other methods, such as interrupted time-series analysis (Anderson *et al.*, 2009, p. 2; Smith and Foxcroft, 2009, p. 3). However, the number of empirical studies available for a systematic review can be small. Smith and Foxcroft's review covers only seven studies and the review by Anderson

et al. covers 13 studies. The survey in the present paper includes 21 studies, with a subsample of 12 studies used for quantitative analysis. While both prior reviews recognize that their conclusions might be contaminated by publication bias, neither goes beyond mere recognition of this problem.² Further, neither review contains a quantitative meta-analysis or presents information regarding joint effects of several types of advertising of alcohol beverages. Hence, these reviews are 'vote counting' exercises, e.g. Anderson *et al.* (2009, p. 13) concluded that '12 of the 13 studies found evidence that such [advertising] exposure predicts both the onset of drinking among non-drinkers and increased level of consumption among existing drinkers'. As demonstrated below, it would be equally correct to state that many studies also found evidence of a null effect for marketing exposure, especially the commercial mass media.

The objective of this paper is to conduct a meta-analysis of longitudinal studies of alcohol marketing and adolescent drinking behaviours, which formally tests for publication bias. I also provide a qualitative evaluation of certain aspects of selection bias. 'Publication bias' was originally defined as the publication or non-publication of empirical results depending on the direction, statistical significance and magnitude of the results (Rothstein *et al.*, 2005).³ Due to emphasis on significance, published studies are likely to be skewed toward larger effects or outcomes, especially when mainstream theory supports a specific effect or there is an overwhelming professional consensus (Ioannidis, 2005; Doucouliagos and Stanley, 2008; Young *et al.*, 2008). If published studies comprise a biased sample of all studies that have been conducted or contain other systematic biases, the results of a literature review or meta-analysis can be misleading. This problem also is known as the 'file drawer problem' because unpublished studies containing insignificant or contradictory results might be found in files maintained by researchers. However, the term 'publication bias' also is used in a broader sense to refer to a number of factors that suppress and distort publication or dissemination of relevant empirical results, including selection biases due to language, availability, cost, familiarity, impact, timing, citation and media coverage (Song *et al.*, 2000; Florax, 2002; Dickersin, 2005; Halpern and Berlin, 2005). The present study includes a qualitative evaluation of dissemination bias in the literature on adolescent drinking. In particular, I provide evidence of selective use of results and outcomes (also known as 'cherry-picking' or 'overreaching') on the part of primary investigators and health policy interest groups.

A study of publication bias in the youth alcohol literature is timely and important for several reasons. First, no prior review in this area, systematic or otherwise, has examined this issue, although several reviews recognize that it may be a problem. Second, traditional narrative reviews often present mixed conclusions with respect to the importance of advertising and marketing, so the two recent surveys are notable for the direction of their conclusions. Third, as noted by Rothstein *et al.* (2005), the problem of selection bias is widespread, so it is imperative for every meta-analysis to include and report an examination of publication bias. The present paper addresses publication bias in order to ensure the integrity of systematic reviews of alcohol marketing and adolescent drinking behaviours. This

is an important step prior to use of longitudinal studies for health policy and related uses.

The remainder of the paper is organized as follows. Section 2 contains a brief narrative review of the longitudinal studies that are included in the meta-analysis. A tabular summary is presented and key features of the studies are described. This section also describes the effect-size data extracted from the primary studies for drinking onset and drinking behaviours by adolescents. Particular problems in the data are addressed, such as the necessity to select a common effect size and the limited number of advertising covariates in some studies. Weighted-mean effect sizes are reported in this section. Section 3 presents a meta-analysis of longitudinal studies of drinking onset, including a funnel plot analysis and meta-regressions that formally test for bias (Egger *et al.*, 1997; Roberts and Stanley, 2005; Stanley, 2005, 2008). Three econometric methods are employed: weighted least squares (WLS), hierarchical multi-level and truncated regressions. Section 4 repeats this analysis for various drinking behaviours. Section 5 discusses qualitative aspects of dissemination bias in the literature on marketing and youth drinking. Selected cross-sectional studies are examined together with the longitudinal studies. Evaluative comments from health policy groups are used to illustrate the bias problem. Section 6 contains the conclusions and recommendations for future research, including policy issues associated with regulation of advertising.

2. Review of Logistic Studies, Data Collection and Weighted Means

This section presents a narrative review of 12 longitudinal studies of alcohol marketing and adolescent alcohol consumption. Many of the studies also are reviewed in Anderson *et al.* (2009) and Smith and Foxcroft (2009), and some details therefore are omitted in this section. A crucial difference with the two earlier reviews is that this section accounts for null (or negative) results for variables that measure alcohol advertising and marketing, which are largely ignored in the two prior reviews. A troublesome problem is the underreporting of empirical results in many longitudinal studies, such as omission of empirical results for all covariates, summary measures of goodness-of-fit and policy forecasts or simulations. This section also presents the data on effect sizes that are employed in the analysis, including fixed- and random-effect weighted means.

2.1 Sample Definition and Data Collection

A first step in a meta-analysis is a literature search and collection of a sample of similar empirical studies that address a particular research question. In the present analysis, the sample is restricted to longitudinal studies of adolescent alcohol use, which contain one or more advertising and marketing variables. Longitudinal studies include a baseline sample and a follow-up sample. Numerous cross-sectional studies of the advertising–drinking relationship therefore are omitted from the formal analysis. A meta-analysis also requires a common effect-size measure that

is contained in the studies or which can be constructed (Nelson and Kennedy, 2009). In the present paper, the analysis is restricted to primary studies that use a logistic-regression model and which report either a log odds ratio or relative risk ratio estimates for one or more marketing variables.⁴ Longitudinal studies that use linear models, multi-level models and other regression formats cannot be combined in a consistent manner. Construction of elasticity estimates also is not possible. The primary studies also must contain information on standard errors or confidence intervals (CIs) for the marketing estimates.⁵ Source materials for the literature search included PubMed, MEDLINE and PsychINFO, with search terms based on descriptors for alcohol drinking, adolescents, youth and various marketing terms such as television, magazines, promotion etc. In addition, after an initial set of longitudinal studies had been identified, an ancestral search was conducted using the Social Science Citation Index (SSCI). A total of 21 longitudinal studies of alcohol marketing and adolescent drinking were identified (a narrative review of all 21 studies is available on the author's web page at <http://econ.la.psu.edu/people>). Logistic models are estimated in 12 of the 21 studies. The two samples of effect sizes are larger because many primary studies include two or more marketing variables.

2.2 *Summary of Primary Studies*

A narrative summary of the 12 primary studies is contained in Table 1, which identifies the study sample, model, marketing variables, positive results and null (or negative) results. Most survey studies use a sample of youthful respondents, ages 16 years or younger. The median sample size is about 1700 participants, ranging from 342 participants in Fisher *et al.* (2007) to 5019 in McClure *et al.* (2009). In some cases, there are two samples analysed, such as boys and girls separately. There are nine US studies, two German studies and one New Zealand study. However, several of the studies use the same data set or extend a prior data set. Two studies use a sample of South Dakota middle school students (Ellickson *et al.*, 2005; Collins *et al.*, 2007) and two studies use a sample of middle school students in New Hampshire and Vermont (McClure *et al.*, 2006; Sargent *et al.*, 2006). Two studies use a sample of German youth (Hanewinkel *et al.*, 2008; Hanewinkel and Sargent, 2009). A common theme in these overlapping studies is use of different drinking measures or emphasis on different methods of alcohol marketing. McClure *et al.* (2006), for example, examine the effects of alcohol-branded merchandise (ABM) on drinking onset, while the study by Sargent *et al.* (2006) uses virtually the same sample and outcome to examine the effects of alcohol portrayals in movies. Neither study mentions the availability of other marketing data, which can lead to omitted variable bias in the reported coefficients. The meta-analysis accounts for the overlap in the primary studies and for possible bias in studies that severely restrict the number of marketing variables. The two systematic reviews ignored these issues. Two of the 12 studies estimate relative risk ratios (Hanewinkel and Sargent, 2009; McClure *et al.*, 2009), but any difference is unimportant because the meta-analysis is conducted using standardized z -statistics for the effect sizes.⁶

Table 1. Summary of Longitudinal Studies of Adolescent Drinking.

| Study, location, survey dates, age groups and sample size | Model and outcome measures | Statistically significant positive results at follow-up | Null and statistically negative results at follow-up |
|--|--|--|---|
| Robinson <i>et al.</i> (1998), San Jose, California, USA, 1994 and 1996, Ages 14/15 and 16/17, $N = 898$ and 635 | Logistic regressions for onset of drinking by baseline non-drinkers and maintenance of drinking by baseline drinkers | Hours per day spent watching TV and hours spent watching music videos are statistically significant for onset of drinking at ages 16/17 by baseline non-drinkers | Null result for use of computer/video games for drinking onset. <i>Negative</i> effect of watching videos in a VCR for drinking onset. Null results for four media variables for maintenance of drinking by baseline drinkers (18-month follow-up) |
| Casswell <i>et al.</i> (2002), Dunedin, New Zealand, 1990, 1993 and 1996, Ages 18, 21 and 26, $N = 714$ | Logistic regression for average number of drinks per occasion and frequency of drinking for males and females separately. Participants assigned to four drinking trajectory groups | Participants at age 18 were asked to rate how much they liked alcohol ads. This variable was never significant for the analysis of drinking trajectories. Access to licensed premises at age 18 had the most consistent impact | Null results for liking of ads for male and female drinkers per occasion and frequency of drinking by trajectory group |
| Stacy <i>et al.</i> (2004), Los Angeles, California, USA, 2000 and 2001, Ages 12/13 and 13/14, $N = 2250$ | Logistic regressions for grade-8 alcohol use (separately for beer and wine/spirits) and 3-drink episodes. Three indexes for TV alcohol ad exposure and two memory tests for ads recall and brand recognition | After adjusting for covariates, the watched TV index is statistically significant for beer use, wine/liquor use and 3-drink episodes. The watched TV sports index is statistically significant for only beer use. Self-reported exposure to TV alcohol ads is significant for beer use | Null results for TV sports index and self-reported exposure for wine/liquor use and 3-drink episodes. Cued-recall memory test is insignificant for all three drinking outcomes. Draw-an-event memory test is insignificant for wine/liquor use and 3-drink episodes, and <i>negative</i> for beer use |

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|--|---|--|--|
| Ellickson <i>et al.</i> (2005), South Dakota, USA, 1997, 1998 and 2000, Ages 12/13 and 14/15, $N = 1206$ and 1905 | Logistic regressions for grade-9 onset of drinking by grade-7 non-drinkers and grade-9 drinking frequency by grade-6 drinkers. Advertising variables obtained at grade 8. Results are corrected based on personal correspondence | For grade-9 drinking onset, results are significant for in-store displays, but the coefficient is improbably large. For grade-9 drinking frequency, significant results for magazines with alcohol ads and beer concession stands at sports and music events. Some results conflict with Collins <i>et al.</i> (2007) for the same sample | For drinking onset, null results for exposure to TV beer ads, magazine ads and beer concession stands. Null results for drinking frequency for TV beer ads and in-store displays. <i>Negative</i> results for weekly TV viewing for both drinking onset and drinking frequency |
| McClure <i>et al.</i> (2006), New Hampshire and Vermont, USA, 1999 and 2000/2001, Ages 10 to 14, $N = 2406$ | Logistic regressions for drinking onset by baseline non-drinkers. Ownership of ABM is determined at follow-up | Ownership of an ABM is significant and positively related to drinking onset. Because ownership of ABMs is measured at follow-up, causality is uncertain | None reported; ownership of an ABM is the only media- and marketing-related variable included in the analysis |
| Sargent <i>et al.</i> (2006), New Hampshire and Vermont, USA, 1999 and 2000/2001, Ages 10 to 14, $N = 2049$ and 357 | Logistic regressions for drinking onset by baseline non-drinkers and prevalence of use by baseline drinkers. Respondents reported exposure to a set of 50 films | Adjusting for covariates, the baseline no. of hours of exposure to alcohol use in movies is a statistically significant predictor of drinking onset and prevalence of use by drinkers | None reported; movie viewing is the only media- or marketing-related variable, adjusted to reflect exposure in the entire sample of 601 movies (assumes proportional exposure with the set of 50 films); median = 8.6 hours of exposure |

Table 1. *Continued.*

| Study, location, survey dates, age groups and sample size | Model and outcome measures | Statistically-significant positive results at follow-up | Null and statistically negative results at follow-up |
|--|---|--|---|
| Collins <i>et al.</i> (2007), South Dakota, USA, 2001 and 2002, Ages 11/12 and 12/13, $N = 1699$ and 1740 | Logistic regressions for grade-7 beer drinking (past year) and drinking intentions, conditional on grade-6 drinking and grade-6 exposure to alcohol ads and marketing | For beer drinking, significant results obtained for sports TV beer ads and ownership of beer promotional items. For grade-7 drinking intentions, significant results for other TV beer ads and promotional items. The coefficient size for promotional items is improbably large for both drinking outcomes. Some results conflict with Ellickson <i>et al.</i> (2005) for the same sample | For beer drinking and intentions, null results for ESPN beer ads, radio listening, magazine reading, concessions, in-store displays and weekly TV viewing. For beer drinking, null results for other TV ads. For intentions, null results for other TV sports beer ads. The authors emphasize the joint effect of the TV ads variables, but this omits the variable for weekly TV viewing |
| Fisher <i>et al.</i> (2007), Nationwide survey, USA, 1996 and 1998/1999, Ages 11 to 18, $N = 5511$ and 261 (postal survey) | Logistic regressions for onset of alcohol use at follow-up and binge drinking by baseline non-drinkers, with separate regressions by gender | For drinking onset, statistically significant results for ownership of (or willingness to use) alcohol promotional items (APIs) for boys and girls. For binge drinking, statistically significant results for APIs for binge drinking by girls | Null results for respondents' awareness of alcohol ads for drinking onset and binge drinking for boys and girls. Null results for APIs for binge drinking by boys |
| Henriksen <i>et al.</i> (2008), Tracy, California, USA, 2003 and 2004, Ages 11–13 and 12–14, $N = 1080$ | Logistic regressions for onset of alcohol use and current drinking by baseline non-drinkers, conditional on alcohol marketing receptivity, brand recognition and brand recall | For drinking onset, significant multivariate results for high level of alcohol marketing receptivity (owned a promotional item such as a hat). For current drinking, significant results for high level of alcohol marketing receptivity | Null results for brand recognition, brand recall and moderate levels of receptivity for onset of drinking and current drinking |

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|--|---|---|--|
| Hanewinkel <i>et al.</i> (2008), Schleswig-Holstein, DE, 2005 and 2006, Ages 10–16 and 11–17, $N = 2110$ | Logistic regression model for onset of binge drinking | The media variable is parental restriction on viewing of FSK-16 movies. Risk ratios are statistically significant for three levels of parental restrictions on viewing of FSK-16 movies (once in a while, sometimes, all) | None reported; restrictions on movie viewing is the only media- or marketing-related variable in the study |
| Hanewinkel and Sargent (2009), Schleswig-Holstein, DE, 2005 and 2006, Ages 10–16 and 11–17, $N = 2708$ | Logistic regressions for drinking onset and binge drinking. Assumes that a participant's exposure in a list of 50 movies is proportional to total exposure in 398 movies | Statistically significant results for hours of exposure to alcohol use in movies, except for moderate level of exposure for binge drinking. Mean exposure time is 3.2 hours | Null results for TV viewing for drinking onset and binge drinking. Null results for moderate movie exposure for binge drinking |
| McClure <i>et al.</i> (2009), Nationwide survey, USA, 2003, 2004 and 2005 (4 waves), Ages 10–14 and 12–16, $N = 4575$ (telephone survey) | Logistic regressions for drinking onset and transition to binge drinking at 8 to 16 months after baseline date and 16 to 24 months. Ownership of ABMs assessed at 8, 16 and 24 months | Trying binge drinking from 8 to 16 months is positively related to ABM ownership at 8 months. Trying alcohol and trying binge drinking from 16 to 24 months are related to change in ABM ownership from 8 to 16 months | Null results for trying alcohol from 8 to 16 months for ABM ownership at 8 months. Null results for trying binge drinking from 16 to 24 months for ABM ownership at 8 months. Exposure to alcohol in movies and exposure to TV are unreported covariates |

Only estimates from multivariate regressions are used in the meta-analysis, although some studies also report bivariate estimates. In general, the practice in this literature is to not report a sensitivity analysis of model specifications. Hence, the problem of multiple estimates for the same covariate from a given study is not encountered. Instead, the problem of interdependence in the meta-analysis is due to estimates for multiple marketing methods from a given regression in each study. For example, Stacy *et al.* (2004) reports results for four different advertising measures for each of three drinking outcomes. The interdependence problem is treated empirically in the analyses by use of cluster robust standard errors and hierarchical mixed-effect models that allow for study-level random errors.

There is broad coverage of methods of alcohol advertising, marketing and promotion. Early empirical studies tended to concentrate on exposure to commercial messages in mass media, especially television. More recent studies employ a wider variety of marketing methods, including in-store displays, branded merchandise and cinema portrayals of alcohol (Ellickson *et al.*, 2005; McClure *et al.*, 2006, 2009; Sargent *et al.*, 2006; Collins *et al.*, 2007; Henriksen *et al.*, 2008). Some studies employ subjective measures of advertising exposure, such as responses to survey questions on 'liking of ads' (Casswell *et al.*, 2002), 'exposure to alcohol ads' (Stacy *et al.*, 2004) and 'brand awareness' (Stacy *et al.*, 2004; Henriksen *et al.*, 2008). All exposure measures are based on self-reports by the respondents, but some are indirect measures of advertising exposure. For example, Robinson *et al.* (1998) used respondents' hours of television viewing as an exposure variable. Similar measures for TV and radio exposure are used in Ellickson *et al.* (2005), Collins *et al.* (2007) and Hanewinkel and Sargent (2009). Finally, market-area variables are omitted covariates in nationwide studies by Fisher *et al.* (2007) and McClure *et al.* (2009), such as market prices, average income, outlet density and various regulations (state monopolies, Sunday closing laws, dry areas). This is another example of misspecification due to omitted variables.

The advertising–marketing variables in Table 1 can be divided into three categories of exposure: mass media advertising, promotion portrayals and other exposures. The three categories are referred to collectively as 'marketing exposure'. It is often argued that some promotion methods, such as ABMs, provide subtle 'cues' with regard to the prevalence or social acceptability of alcohol that adversely affect adolescents (Sargent *et al.*, 2006; Wills *et al.*, 2009). Hence, the meta-analysis distinguishes between exposure to conventional mass media (e.g. TV, radio, magazines), promotion portrayals (ABMs, movies, videos) and other exposures (in-store displays, concessions, games). All subjective measures (e.g. liking of ads, brand recall) also are placed in the 'other' category. The number of marketing covariates range from nine in Collins *et al.* (2007) to only one variable in several studies. One covariate might be acceptable if it is a broad index, such as 'liking of ads', but some studies report empirical results for only ABMs or movies (McClure *et al.*, 2006, 2009; Sargent *et al.*, 2006; Hanewinkel *et al.*, 2008; Henriksen *et al.*, 2008). Studies with one or two measures are especially suspect for bias due to misspecification of the regression model and tend to produce effect-size estimates that are larger in magnitude compared to studies for the mass media. Further,

the regression specifications in Table 1 range from simple models with a few covariates (Robinson *et al.*, 1998) to elaborate models with 20 or more explanatory variables (Stacy *et al.*, 2004; Hanewinkel and Sargent, 2009). A summary of the covariates used in longitudinal studies appears in Anderson *et al.* (2009). The non-advertising covariates fall into four categories: demographics (age, gender, race, parenting); social influences to drink (peers' drinking, parents' drinking, religiosity, alcohol access, baseline drinking); personality traits (self esteem, rebelliousness, risk taking, smoking status) and other influences (school performance, sports participation). Omission of personality traits is likely to create specification bias, which is accounted for in the meta-analysis.

The results for each primary study for the advertising and marketing exposure variables are summarized in Table 1. All of the studies contain at least one statistically significant positive coefficient for all participants or an age/gender cohort. With three exceptions, all of the studies contain at least one null result. The exceptions are McClure *et al.* (2006), Sargent *et al.* (2006) and Hanewinkel *et al.* (2008), which report exposure for only one promotional method. In general, the longitudinal studies contain a wide variety of empirical results that could be used to support or refute claims of adverse effects due to alcohol advertising. Given the requirement of a common effect size, drinking outcomes in the meta-analysis are divided into two categories: drinking onset by baseline non-drinkers; and other drinking behaviours at follow-up (drinking maintenance, frequency, amount, binge drinking).

2.3 Meta-sample and Effect-size Means for Drinking Onset

The first meta-sample consists of eight empirical studies that contain 23 estimates of the effects of marketing exposure on adolescent drinking onset (initiation). Only seven of the studies are independent because McClure *et al.* (2006) and Sargent *et al.* (2006) use virtually identical samples of New England youth. The effect-size data for these 23 estimates are shown in Table 2. Measures of alcohol advertising and marketing in the table include TV viewing, magazines, in-store displays, beer concession stands, branded merchandise and movie/video viewing. Most empirical studies measure exposure on a continuous scale, but some studies use a set of binary variables. The intent in these studies is to represent a non-linear relationship, but this also produces interdependent effect-size estimates and data outliers. In order to avoid these problems, I used only one estimate from Hanewinkel and Sargent (2009).⁷ Some estimates are adjusted marginally for rounding errors in the CI estimates.

Estimates of the fixed-effect mean for drinking onset are shown at the bottom of Table 2. Due to the small sample size, mass media and other exposures are combined into a single category. For all 23 estimates, the fixed-effect mean for the odds ratio is 1.099 (95% CI, 1.064–1.136; $p < 0.001$), which is statistically significant but small in magnitude.⁸ The fixed-effect mean assigns greater weight to more precise estimates. When less precise studies are given greater weight, the random-effect and unweighted means show larger values. This result is consistent

Table 2. Drinking Onset: Meta-analysis Data and Means.

| Study | Drinking outcome | Media exposure | Odds ratio (95% CI) | Log odds ratio (se) | z-statistic (p-value) | Sample size (df) |
|--------------------------------|-----------------------------|-------------------------|---------------------------|------------------------|------------------------|------------------|
| <i>Mass media advertising</i> | | | | | | |
| Robinson <i>et al.</i> (1998) | Drinking onset | TV viewing | 1.090 (1.01, 1.18) | 0.0862 (0.040) | 2.1715 (0.030) | 898 (889) |
| Ellickson <i>et al.</i> (2005) | Drinking onset | TV beer ads | 1.050 (0.64, 1.70) | 0.0488 (0.249) | 0.1958 (0.845) | 1206 (1179) |
| Ellickson <i>et al.</i> (2005) | Drinking onset | Weekly TV viewing | 0.780 (0.69, 0.88) | -0.2485 (0.062) | -4.0042 (0.000) | 1206 (1179) |
| Hanewinkel and Sargent (2009) | Drinking onset | Hours TV viewing | 0.990 (0.75, 1.31) | -0.0101 (0.142) | -0.0706 (0.944) | 2708 (2685) |
| Ellickson <i>et al.</i> (2005) | Drinking onset | Magazines with ads | 1.120 (0.94, 1.30) | 0.1044 (0.083) | 1.2617 (0.207) | 1206 (1179) |
| <i>Promotional portrayals</i> | | | | | | |
| McClure <i>et al.</i> (2006) | Drinking onset | Alcohol merchandise | 1.500 (1.10, 2.00) | 0.4055 (0.152) | 2.6586 (0.008) | 2406 (2390) |
| Fisher <i>et al.</i> (2007) | Drinking onset – boys | Alcohol merchandise | 1.780 (1.36, 2.33) | 0.5766 (0.137) | 4.1983 (0.000) | 2228 (2210) |
| Fisher <i>et al.</i> (2007) | Drinking onset – girls | Alcohol merchandise | 1.740 (1.37, 2.19) | 0.5539 (0.120) | 4.6285 (0.000) | 3283 (3265) |
| Henriksen <i>et al.</i> (2008) | Drinking onset | Alcohol merchandise | 1.200 (0.75, 1.90) | 0.1823 (0.237) | 0.7689 (0.442) | 1080 (1065) |
| Henriksen <i>et al.</i> (2008) | Drinking onset | Alcohol merchandise | 1.680 (1.20, 2.35) | 0.5188 (0.172) | 3.0258 (0.002) | 1080 (1065) |
| McClure <i>et al.</i> (2009) | Drink onset at 8–16 months | Alcohol merchandise | 1.410 (0.98, 2.01) | 0.3436 (0.183) | 1.8750 (0.061) | 4575 (4558) |
| McClure <i>et al.</i> (2009) | Drink onset at 16–24 months | Alcohol merchandise | 1.570 (0.99, 2.50) | 0.4511 (0.236) | 1.9088 (0.056) | 4575 (4558) |
| Sargent <i>et al.</i> (2006) | Drinking onset | Hours movie alcohol use | 1.150 (1.06, 1.25) | 0.1398 (0.042) | 3.3229 (0.001) | 2406 (2388) |
| Hanewinkel and Sargent (2009) | Drinking onset | Hours movie alcohol use | 1.420 (1.16, 1.75) | 0.3507 (0.105) | 3.3428 (0.001) | 2708 (2685) |
| Robinson <i>et al.</i> (1998) | Drinking onset | Music TV videos | 1.310 (1.17, 1.47) | 0.2700 (0.058) | 4.6372 (0.000) | 898 (889) |
| Robinson <i>et al.</i> (1998) | Drinking onset | VCR videos | 0.890 (0.79, 0.99) | -0.1278 (0.058) | -2.2205 (0.026) | 898 (889) |

| Other media and brand awareness | | | | | | |
|--|------------------------|------------------------|-----------------------------|-----------------------|-----------------------|-------------|
| Robinson <i>et al.</i> (1998) | Drinking onset | Computer games | 0.940 (0.84, 1.05) | −0.0619 (0.057) | −1.0870 (0.277) | 898 (889) |
| Ellickson <i>et al.</i> (2005) | Drinking onset | Beer concessions | 1.060 (0.83, 1.40) | 0.0677 (0.133) | 0.5073 (0.612) | 1206 (1179) |
| Ellickson <i>et al.</i> (2005) | Drinking onset | In-store displays | 1.420 (1.10, 1.80) | 0.3507 (0.126) | 2.7911 (0.005) | 1206 (1179) |
| Fisher <i>et al.</i> (2007) | Drinking onset – boys | Awareness of ads | 1.270 (0.98, 1.64) | 0.2390 (0.131) | 1.8196 (0.069) | 2228 (2210) |
| Fisher <i>et al.</i> (2007) | Drinking onset – girls | Awareness of ads | 1.040 (0.84, 1.29) | 0.0392 (0.109) | 0.3584 (0.720) | 3283 (3265) |
| Henriksen <i>et al.</i> (2008) | Drinking onset | Beer brand recognition | 1.070 (0.93, 1.23) | 0.0677 (0.071) | 0.9486 (0.343) | 1080 (1065) |
| Henriksen <i>et al.</i> (2008) | Drinking onset | Beer brand recall | 1.100 (0.97, 1.25) | 0.0953 (0.065) | 1.4732 (0.141) | 1080 (1065) |
| Fixed-effect mean – all | | | 1.099 (1.064, 1.136) | 0.0947 (0.017) | 5.7044 (0.000) | |
| Fixed-effect mean – mass media and other media | | | 1.032 (0.989, 1.077) | 0.0312 (0.022) | 1.4331 (0.152) | |
| Fixed-effect mean – promotion portrayals | | | 1.201 (1.142, 1.263) | 0.1830 (0.026) | 7.1302 (0.000) | |

Notes: Bold entries indicate statistically significant estimates, 95% confidence level. Two values are significantly negative. Random-effects means (*p*-value) for the odds ratio are 1.178 (0.000), 1.051 (0.265) and 1.364 (0.000). Unweighted means (sd) are 1.243 (0.278), 1.078 (0.158) and 1.423 (0.273). All calculations and weighted means computed using CMA2.2 (Borenstein *et al.*, 2008).

with publication bias, but also can reflect study heterogeneity. However, only nine of 23 estimates (39%) are significantly positive and eight of these estimates are larger than the upper-CI limit of 1.136. Only one estimate for mass media (Robinson *et al.*, 1998) is statistically positive. Several studies produce point estimates that seem improbably large, including two estimates by Fisher *et al.* (2007) and one estimate by Henriksen *et al.* (2008) for ABMs. The fixed-effect mean for 12 estimates for mass media and other exposures is 1.032 ($p = 0.152$), which is not statistically significant. When the sample is restricted to 11 estimates for ABMs and movies/videos, the mean is 1.201 ($p < 0.001$). Hence, the analysis suggests a positive association for promotion portrayals, but not for mass media and other exposures. In general, the data in Table 2 indicate a non-robust effect of marketing on drinking onset, but exposure to branded merchandise and movie/video portrayals might be cause for concern. Omitted variable bias also is an issue in the promotion studies.

2.4 *Meta-sample and Effect-size Means for Drinking Behaviours*

The second meta-sample consists of nine empirical studies that contain 40 estimates of the effect of advertising and marketing on adolescent drinking behaviours. These estimates are shown in Table 3. Only eight of the studies are independent because Hanewinkel *et al.* (2008) and Hanewinkel and Sargent (2009) use similar samples for German youth. A variety of drinking behaviours are examined, including maintenance of drinking by baseline drinkers, drinking amount by beverage, binge drinking and onset of binge drinking. Measures of advertising and marketing exposure include TV viewing, magazine reading, radio listening, in-store displays, beer concession stands, brand recall, branded merchandise and movie/video viewing. Adjustments to the data include accounting for rounding errors in the estimates of the CIs and the use of only two estimates from Hanewinkel and Sargent (2009) and one estimate from Hanewinkel *et al.* (2008). These exclusions again reflect the use of a set of binary variables for promotion portrayals.

Estimates of the fixed-effect mean for drinking behaviours are reported at the bottom of Table 3. The fixed-effect mean for the odds ratio is 1.103 (95% CI, 1.074–1.132; $p < 0.001$), which is significant but modest in magnitude. However, only 12 of 40 estimates (30%) are significantly positive and all of these estimates are larger than the upper-CI limit of 1.132. This result is consistent with publication bias. Only five of 14 estimates for mass media are statistically significant, but four of these estimates are from the study by Stacy *et al.* (2004) for youth in the Los Angeles area. Six of 12 estimates for promotion are significant, but several z -statistics are close to the lower limit of 2.0. Several studies produce point estimates that seem improbably large, including estimates for ABMs in Collins *et al.* (2007), Fisher *et al.* (2007) and McClure *et al.* (2009), and the estimate for movie portrayals in Hanewinkel and Sargent (2009). When less precise studies are given greater weight, the random-effect and unweighted means show larger values. Again, this

Table 3. Drinking Behaviours: Meta-analysis Data and Means.

| Study | Drinking behaviour | Media exposure | Odds ratio (95% CI) | Log odds ratio (se) | z-stat (p-value) | Sample size (df) |
|---------------------------------|--------------------------------|--------------------------|---------------------------|------------------------|------------------------|---------------------|
| <i>Mass media advertising</i> | | | | | | |
| Robinson <i>et al.</i> (1998) | Drinking maintenance | TV viewing | 1.010 (0.93, 1.11) | 0.0100 (0.043) | 0.2323 (0.816) | 635 (626) |
| Stacy <i>et al.</i> (2004) | Beer drinking | TV ads | 1.440 (1.27, 1.61) | 0.3646 (0.058) | 6.2316 (0.000) | 2250 (2230) |
| Stacy <i>et al.</i> (2004) | Beer drinking | TV sports ads | 1.200 (1.05, 1.37) | 0.1823 (0.068) | 2.6866 (0.007) | 2250 (2230) |
| Stacy <i>et al.</i> (2004) | Wine/liquor drinking | TV ads | 1.340 (1.17, 1.52) | 0.2927 (0.067) | 4.3837 (0.000) | 2250 (2230) |
| Stacy <i>et al.</i> (2004) | Wine/liquor drinking | TV sports ads | 1.000 (0.88, 1.15) | 0.0010 (0.068) | 0.0000 (1.000) | 2250 (2230) |
| Stacy <i>et al.</i> (2004) | 3-drink episodes | TV ads | 1.260 (1.08, 1.48) | 0.2311 (0.080) | 2.8753 (0.004) | 2249 (2229) |
| Stacy <i>et al.</i> (2004) | 3-drink episodes | TV sports ads | 1.070 (0.91, 1.26) | 0.0677 (0.083) | 0.8150 (0.415) | 2249 (2229) |
| Collins <i>et al.</i> (2007) | Beer drinking | ESPN-TV beer ads | 1.080 (0.83, 1.42) | 0.0770 (0.137) | 0.5618 (0.574) | 1699 (1673) |
| Collins <i>et al.</i> (2007) | Beer drinking | TV sports beer ads | 1.190 (1.01, 1.40) | 0.1740 (0.083) | 2.0883 (0.037) | 1699 (1673) |
| Collins <i>et al.</i> (2007) | Beer drinking | Other TV beer ads | 0.130 (0.95, 1.34) | 0.1222 (0.088) | 1.3928 (0.164) | 1699 (1673) |
| Collins <i>et al.</i> (2007) | Beer drinking | TV viewing | 0.860 (0.73, 1.03) | -0.1508 (0.095) | 1.5891 (0.112) | 1699 (1673) |
| Hanewinkel and Sargent (2009) | Binge drinking | Hours TV viewing | 0.760 (0.48, 1.19) | -0.2744 (0.232) | -1.1849 (0.236) | 2708 (2685) |
| Collins <i>et al.</i> (2007) | Beer drinking | Magazine reading | 0.960 (0.87, 1.06) | -0.0408 (0.050) | -0.8101 (0.418) | 1699 (1673) |
| Collins <i>et al.</i> (2007) | Beer drinking | Radio listening | 1.170 (1.00, 1.37) | 0.1570 (0.080) | 1.9550 (0.051) | 1699 (1673) |
| <i>Promotional portrayals</i> | | | | | | |
| Collins <i>et al.</i> (2007) | Beer drinking | Beer merchandise | 1.760 (1.23, 2.52) | 0.5653 (0.183) | 3.0896 (0.002) | 1699 (1673) |
| Fisher <i>et al.</i> (2007) | Binge drinking, boys | Alcohol merchandise | 0.870 (0.51, 1.48) | -0.1393 (0.272) | -0.5124 (0.608) | 342 (324) |
| Fisher <i>et al.</i> (2007) | Binge drinking, girls | Alcohol merchandise | 1.790 (1.16, 2.77) | 0.5822 (0.222) | 2.6220 (0.009) | 563 (545) |
| Henriksen <i>et al.</i> (2008) | Current drinking | Receptive to merchandise | 1.190 (0.62, 2.26) | 0.1740 (0.330) | 0.5272 (0.598) | 903 (886) |
| Henriksen <i>et al.</i> (2008) | Current drinking | Receptive to merchandise | 1.620 (1.01, 2.60) | 0.4824 (0.241) | 2.0000 (0.0455) | 903 (886) |
| McClure <i>et al.</i> (2009) | Binge drinking at 8–16 months | Alcohol merchandise | 1.800 (1.28, 2.54) | 0.5878 (0.175) | 3.3621 (0.001) | 5019 (5002) |
| McClure <i>et al.</i> (2009) | Binge drinking at 16–24 months | Alcohol merchandise | 1.440 (0.90, 2.31) | 0.3646 (0.240) | 1.5164 (0.129) | 4575 (4558) |
| Hanewinkel <i>et al.</i> (2008) | Binge drinking | Parents limit movies | 1.640 (1.03, 2.63) | 0.4947 (0.239) | 2.0686 (0.039) | 2110 (2097) |
| Hanewinkel and Sargent (2009) | Binge drinking | Hours movie viewing | 1.440 (0.96, 2.17) | 0.3646 (0.208) | 1.7527 (0.080) | 2708 (2685) |
| Hanewinkel and Sargent (2009) | Binge drinking | Hours movie viewing | 1.950 (1.27, 3.00) | 0.6678 (0.219) | 3.0454 (0.002) | 2708 (2685) |

Table 3. *Continued.*

| Study | Drinking behaviour | Media exposure | Odds ratio (95% CI) | Log odds ratio (se) | z-stat (<i>p</i> -value) | Sample size (df) |
|--|-----------------------|-------------------------|-----------------------------|------------------------|---------------------------|---------------------|
| Robinson <i>et al.</i> (1998) | Drinking maintenance | Music TV videos | 1.050 (0.95, 1.17) | 0.0488 (0.053) | 0.9182 (0.358) | 635 (626) |
| Robinson <i>et al.</i> (1998) | Drinking maintenance | VCR videos | 0.970 (0.86, 1.10) | -0.0305 (0.063) | -0.4851 (0.628) | 635 (626) |
| <i>Other media and brand awareness</i> | | | | | | |
| Robinson <i>et al.</i> (1998) | Drinking maintenance | Computer games | 1.000 (0.89, 1.12) | 0.0000 (0.059) | 0.0000 (1.000) | 635 (626) |
| Collins <i>et al.</i> (2007) | Beer drinking | Beer concessions | 1.010 (0.91, 1.13) | 0.0100 (0.055) | 0.1801 (0.857) | 1699 (1673) |
| Collins <i>et al.</i> (2007) | Beer drinking | In-store beer ads | 1.030 (0.92, 1.14) | 0.0296 (0.057) | 0.5193 (0.604) | 1699 (1673) |
| Casswell <i>et al.</i> (2002) | Higher trajectory | Liking of ads | 1.600 (0.96, 2.70) | 0.4700 (0.264) | 1.7817 (0.075) | 407 (401) |
| Stacy <i>et al.</i> (2004) | Beer drinking | Self-report ad exposure | 1.210 (1.04, 1.41) | 0.1906 (0.078) | 2.4550 (0.014) | 2250 (2230) |
| Stacy <i>et al.</i> (2004) | Wine/liquor drinking | Self-report ad exposure | 1.180 (0.98, 1.32) | 0.1655 (0.091) | 1.8190 (0.069) | 2250 (223) |
| Stacy <i>et al.</i> (2004) | 3-drink episodes | Self-report ad exposure | 1.060 (0.89, 1.27) | 0.0583 (0.091) | 0.6424 (0.521) | 2249 (2229) |
| Stacy <i>et al.</i> (2004) | Beer drinking | Brand recall | 1.170 (0.97, 1.38) | 0.1570 (0.092) | 1.7107 (0.087) | 2250 (2230) |
| Stacy <i>et al.</i> (2004) | Wine/liquor drinking | Brand recall | 1.070 (0.91, 1.26) | 0.0677 (0.083) | 0.8150 (0.415) | 2250 (2230) |
| Stacy <i>et al.</i> (2004) | 3-drink episodes | Brand recall | 1.170 (0.91, 1.44) | 0.1570 (0.109) | 1.4429 (0.149) | 2249 (2229) |
| Henriksen <i>et al.</i> (2008) | Current drinking | Beer brand recognition | 1.130 (0.93, 1.38) | 0.1222 (0.101) | 1.2139 (0.225) | 903 (886) |
| Henriksen <i>et al.</i> (2008) | Current drinking | Beer brand recall | 1.110 (0.94, 1.33) | 0.1044 (0.088) | 1.1787 (0.238) | 903 (886) |
| Fisher <i>et al.</i> (2007) | Binge drinking, boys | Awareness of ads | 0.980 (0.58, 1.66) | -0.0202 (0.272) | -0.0753 (0.940) | 342 (324) |
| Fisher <i>et al.</i> (2007) | Binge drinking, girls | Awareness of ads | 1.160 (0.77, 1.74) | 0.1484 (0.208) | 0.7136 (0.475) | 563 (545) |
| Fixed-effect mean – all media | | | 1.103 (1.074, 1.132) | 0.0979 (0.013) | 7.3032 (0.000) | |
| Fixed effect mean – mass media | | | 1.111 (1.070, 1.153) | 0.1051 (0.019) | 5.5611 (0.000) | |
| Fixed-effect mean – promotion portrayals | | | 1.135 (1.060, 1.216) | 0.1270 (0.035) | 3.6119 (0.000) | |
| Fixed-effect mean – other media | | | 1.078 (1.032, 1.127) | 0.0755 (0.023) | 3.3434 (0.001) | |

Notes: Bold entries indicate statistically significant estimates, 95% confidence level. Random-effects means (*p*-value) for the odds ratio are 1.140 (0.000), 1.113 (0.012), 1.379 (0.000) and 1.078 (0.001). Unweighted means (sd) are 1.222 (0.285), 1.050 (0.183), 1.460 (0.362) and 1.134 (0.154). All calculations and weighted means computed using CMA2.2 (Borenstein *et al.*, 2008).

could be due to publication bias. When the sample is restricted to mass media, the fixed-effect mean is 1.111 (1.070–1.132; $p < 0.001$). For promotion, the mean is 1.135 (1.060–1.216; $p < 0.001$). For other exposures, the mean is only 1.078 (1.032–1.127; $p = 0.001$). Compared to drinking onset, the array of data in Table 3 suggests a more robust association of marketing with adolescent alcohol behaviours. However, as pointed out by Smith and Foxcroft (2009), inferences about modest effect sizes are limited by the potential influence of unmeasured confounders. The meta-regression analysis attempts to sort out this heterogeneity for drinking onset and drinking behaviours.

The studies in Tables 2 and 3 present some significant effect-size estimates and an even larger number of insignificant estimates. Overall, there are 63 estimates, which are statistically significant in only 21 cases (33%). The tables contain 16 estimates of the effect of TV viewing, which are significantly positive in six cases and insignificant (or negative) in 10 cases. Both estimates for magazine advertisements are insignificant. There are 14 estimates for ABMs, eight of which are significantly positive and six are insignificant. A similar problem exists for studies of movie portrayals of alcohol. Finally, there are 15 estimates for subjective measures of 'awareness of ads', 'liking of ads', 'brand recall' and 'self-reported ad exposure', and only one effect-size estimate is significant. This raises a question of what exactly is being captured by supposedly objective measures of marketing exposure. One possibility is that youth who are predisposed to drink for other reasons also are attracted to advertising and marketing, which might be captured by personality traits. However, in order to sort out this influence, more complex surveys are required that trace marketing exposure, personality development and drinking behaviours over a longer time period. The New Zealand studies fall into this model, but these contain very few significant effects. For example, Connolly *et al.* (1994) reported significantly positive results for only three out of 48 advertising coefficients. More generally, longitudinal studies need to treat advertising and marketing exposure as an endogenous variable. Hence, a basic problem is that conditions for demonstrating causality are unlikely to be satisfied, despite the use of prospective data (Geweke and Martin, 2002; Heckman *et al.*, 2008; Nelson, 2010b). The two systematic reviews ignored these issues.

3. Publication Bias in Drinking Onset Studies

This section analyses publication bias in the sample of 23 estimates for the effect of alcohol marketing on drinking onset by adolescents. There are four steps in the analysis: first, graphical analyses of log odds ratios are presented using funnel plots that account for 'missing' studies. The second step estimates bivariate regressions for the standard normal deviates or z -statistics. The third step is a multivariate meta-regression analysis that incorporates covariates for study characteristics and publication selection. The purpose of the multivariate analysis is to simultaneously account for study heterogeneity and publication bias. The fourth step is to estimate truncated regression models that might better represent the underlying population.

The funnel plots reveal information about the missing part of the data, which serves as a basis for the lower limit in the truncated regressions. Because there is interdependence or clustering among the estimates, it also is necessary to account for this feature of the data. Two methods are considered: first, cluster robust standard errors; and second, a random-effects multi-level (REML) regression, which is estimated by restricted maximum likelihood. All regressions are corrected for heteroskedasticity by weighting by the inverse of the standard error. Definitions for explanatory variables are reported below and also appear in the regression tables.

3.1 *Filled Funnel Plots*

Two funnel plots are shown in Figure 1, where log odds ratios are plotted against standard errors (upper panel) and inverse of the standard errors or precision (lower panel).⁹ The filled funnel plots, computed using the CMA2.2 software package (Borenstein *et al.*, 2008), show the actual observations from Table 2 and imputed values (filled dots) obtained using the trim-and-fill procedure (Duval and Tweedie, 2000; Duval, 2005). Funnel plots can be difficult to interpret, especially when the number of observations is small. The non-parametric trim-and-fill procedure imputes missing observations necessary for symmetry and recomputes the combined effect size, which facilitates detection of publication bias. There are eight imputed observations and the recomputed fixed-effect mean is only 1.053 (95% CI, 1.021–1.086), shown by a filled diamond on the horizontal axis and vertical line in Figure 1. The recomputed random-effects mean is 1.054 (0.968–1.148), which is not significant. In the absence of publication bias, plots of the actual data are symmetric about the mean effect size, which is shown by a clear diamond on the horizontal axis. In the presence of bias, there will be a higher concentration of observations on one side of the mean. This reflects the notion that less precise studies are more likely to be published if they have larger than average effects, which makes them more likely to meet criteria for statistical significance or contain important public policy implications. Positive-bias asymmetry appears as a gap in the lower left-hand portion of the funnel plots. Hence, the asymmetric plots in Figure 1 provide evidence of publication bias due to omission of less precise estimates with small or negative odds ratios. The plots also suggest the lack of a genuine effect or at least a smaller mean effect size. The implication is that some studies are either unpublished or that published studies select results to emphasize larger positive results. Note that negative values are not required for a finding of no publication bias, only symmetric distribution of the actual observations about the fixed-effect mean. Lastly, asymmetry also can arise for reasons other than publication bias, such as heterogeneity due to study methodology, sampling errors and genuine differences in population effect sizes across media, time or study area.

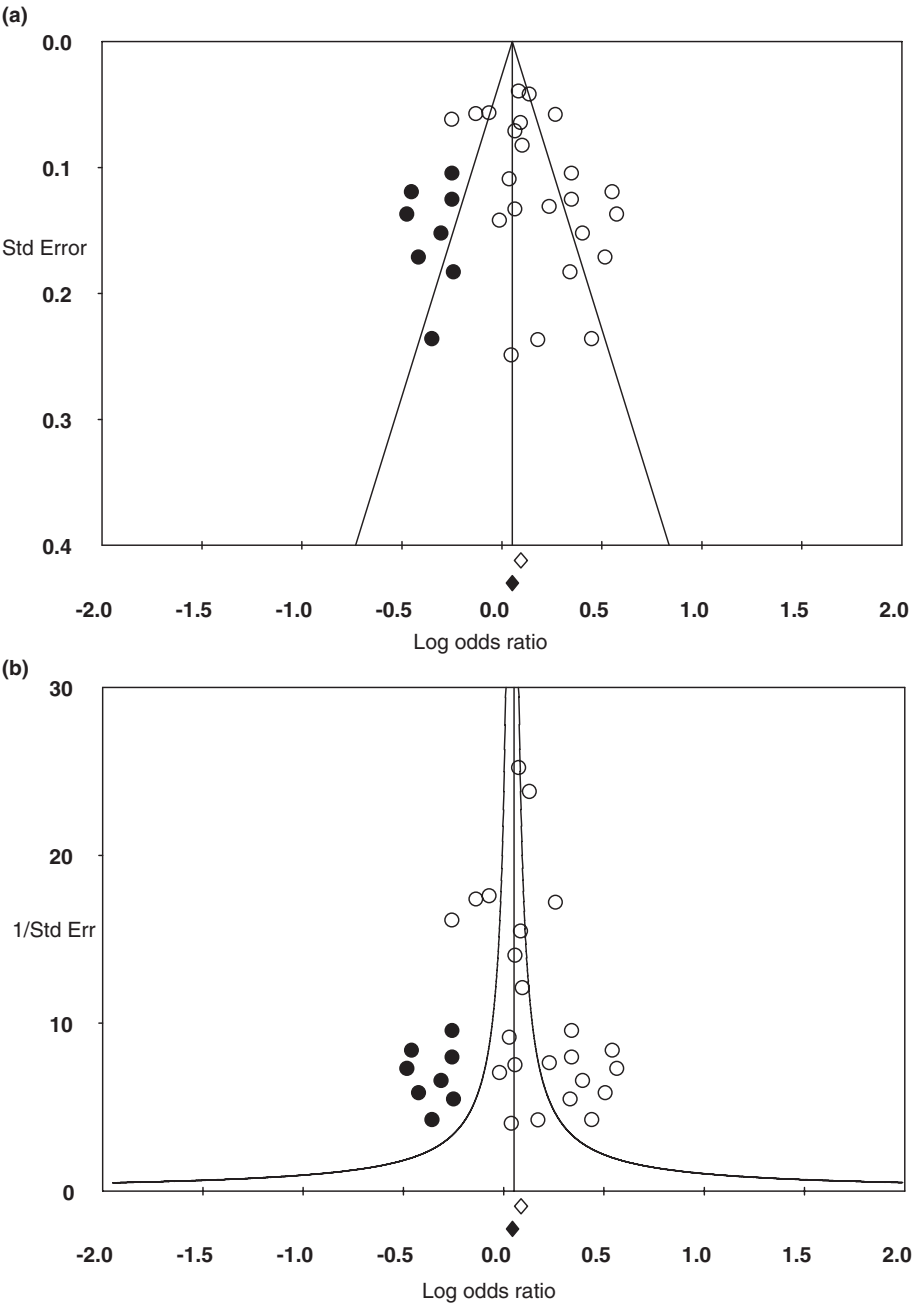


Figure 1. (a) Funnel Plot of Standard Error, Drinking Onset. (Filled dots are imputed studies; diamonds are means.) (b) Funnel Plot of Precision, Drinking Onset. (Filled dots are imputed studies; diamonds are means.)

3.2 Bivariate Regression Model

Using the fixed-effect meta-analysis model, the effect size in the i th primary study can be represented as (Card and Kruger, 1995; Stanley, 2005, 2008)

$$ES_i = \beta_1 + \beta_0 (Se_i) + \varepsilon_i \quad (1)$$

where ES is the estimated effect size (log odds ratio), Se is its estimated standard error and ε is a stochastic error term. In the absence of selection and heterogeneity, observed effects should vary randomly about the true effect size, β_1 , independent of the standard error. However, if specifications and estimates are selected based on the significance of the main covariates, selection bias will vary directly with the standard error, i.e. larger Se values are associated generally with larger effect-size estimates. Because the estimates are inherently heteroskedastic, it is appropriate to divide equation (1) by the standard error to yield (Egger *et al.*, 1997; Stanley, 2005; Sterne and Egger, 2005)

$$z_i = \beta_0 + \beta_1(1/Se_i) + v_i \quad (2)$$

where z is the standard normal deviate or z -statistic, $1/Se$ is its precision and v is an error term. Equation (2) is equivalent to a WLS regression of the effect size on its standard error, with inverse variance weights (Sterne and Egger, 2005). Hence, equation (2) is referred to as the 'Egger intercept test' or, alternatively, the funnel-graph asymmetry test (FAT); see Sterne and Egger (2005) and Stanley (2005, 2008).¹⁰ The null hypothesis of no asymmetry implies an insignificant intercept estimate. If the null is rejected, the magnitude and direction of asymmetry is indicated by the intercept. A positive intercept indicates that there is a concentration of observations in the lower-right hand portion of the funnel plot. Further, the slope estimate in equation (2) indicates the effect size after removing the influence of asymmetry, and a significance test for the slope is referred to as the precision-effect test (PET). An insignificant slope is consistent with lack of a genuine effect (Stanley, 2008). Accounting for interdependence in the standard errors, Table 4 shows results for the FAT–PET bivariate tests for drinking onset. In column (1) the WLS intercept has a significantly positive value of 1.984 ($p = 0.011$), which is substantial.¹¹ The slope estimate of -0.044 ($p = 0.566$) is not significantly different from zero, suggesting the absence of a genuine effect of marketing exposure. Column (4) shows the bivariate results using the REML model, where again the intercept is significantly positive and the precision slope is insignificant.

3.3 Multivariate Meta-regression Analysis Model

The low R^2 value in column (1) is an indicator of heterogeneity among the sample of estimates for drinking onset, which might be explainable using a multivariate model. Further, both the Q -test and the I^2 test reject homogeneity in the data ($Q = 117.3$, $p < 0.001$; $I^2 = 81.2$, $p < 0.001$). In order to account for heterogeneity, the multivariate analysis uses a meta-regression model due to Doucouliagos and Stanley (2009). Their model expands the Egger model in equation (2) to account for

Table 4. Meta-regressions for Drinking Onset and Publication Bias.

| Variables | (1) WLS | (2) WLS | (3) WLS | (4) REML | (5) REML | (6) REML | (7) Truncate | (8) Truncate | (9) Truncate |
|-------------------------|------------------------|------------------------|-------------------------|------------------------|------------------------|------------------|------------------------|------------------------|-------------------------|
| Intercept | 1.984 (3.80) | 1.882 (3.91) | 3.130 (4.92) | 1.988 (2.12) | 1.859 (2.08) | 3.126 (1.34) | 1.976 (3.74) | 1.894 (3.97) | 3.175 (5.58) |
| Precision | -0.044 (0.96) | -0.097 (1.59) | -0.020 (0.21) | -0.044 (0.59) | -0.093 (1.25) | -0.021 (0.24) | -0.046 (0.96) | -0.102 (1.64) | -0.022 (0.24) |
| Promotion portrayal wt. | | 0.144 (1.87) | 0.129 (1.85) | | 0.140 (2.08) | 0.129 (1.75) | | 0.148 (2.00) | 0.132 (2.12) |
| Ads covariate binary | | | 1.270 (2.12) | | | 1.251 (1.21) | | | 1.292 (2.28) |
| SSCI impact factor | | | -0.590 (2.52) | | | -0.586 (0.95) | | | -0.607 (2.82) |
| Std errors | cluster | cluster | cluster | multi | multi | multi | cluster | cluster | cluster |
| R^2 | 0.016 | 0.202 | 0.291 | — | — | — | — | — | — |
| N | 23 | 23 | 23 | 23 | 23 | 23 | 23 | 23 | 23 |

Notes: T - and z -statistics (absolute values) in parentheses; bold entries indicate significant coefficients at the 95% level. WLS regressions with cluster robust standard errors based on study id. REML are multi-level mixed-effects regressions, with standard errors based on restricted maximum likelihood estimation. Maximum-likelihood truncated regressions are restricted to minimum effect size or larger, with cluster robust standard error based on study id. All estimates obtained using Stata/IC 10.1.

Variables: Dependent variable is the z -statistic for the log odds ratio; Precision is the inverse of the standard error; Promotion portrayals is a binary variable for branded merchandise and movie exposure, weighted by the inverse standard error; Ads covariate is a binary variable if the study has one or two variables for advertising and marketing exposure; and SSCI impact factor is the ISI-JCR measure of journal quality (see Murtaugh, 2002).

two types of variables. First, heterogeneity is represented by a set of M moderators that explain methodological variation in log odds ratios in Table 2, such as the type of media or type of drinking behaviour. Second, a set of K variables is assumed to be correlated with the publication selection process, such as the number of advertising covariates or journal quality. The inclusion of the M variables is a standard procedure in meta-regression analyses, so it is the K variables that are novel and reveal what guides the selection process, other things being held constant. The multivariate meta-regression analysis (MRA) model for publication bias can be written as (Doucouliagos and Stanley, 2009)

$$z_i = \beta_0 + \beta_1(1/Se_i) + \sum \alpha_m(M_{im}/Se_i) + \sum \gamma_k K_{ik} + v_i \quad (3)$$

where the M variables are divided by the standard error to correct for heteroskedasticity and the K variables are not. Publication selection is now captured by a combination of the K variables and the intercept term. Individual coefficients indicate the direction and magnitude of bias due to each of the K variables. Genuine effects are captured by combinations of the M variables (i.e. those variables divided by Se) and the precision term.

The data set for drinking onset does not classify as exceptionally rich as most of the primary studies follow similar methodologies. It contains 23 observations from only seven independent studies and, given the structure of the data, most moderators are defined at the study level; i.e. no variation is present at the level of the individual estimates other than the type of advertising or marketing.¹² In light of this restriction, the specification of equation (3) for drinking onset is a simple extension of equation (2). The M variable is the use of promotion media as a measure of alcohol marketing. The K variables are a binary variable for the number of advertising covariates (=1 if the number of covariates is two or less) and the SSCI impact factor for journal quality (see Murtaugh, 2002). With these additional results, the intercepts in Table 4 are significantly positive in columns (2) and (3) for WLS, and in column (5) for REML. The WLS binary covariate for ads is significantly positive in column (3) and the SSCI impact factor is significantly negative. The increase in the R^2 values justifies the use of an MRA model.

3.4 *Truncated Regression Model for Drinking Onset*

In the presence of publication bias, a sample of effect sizes is a restricted set of all relevant results. That is, entire observations are missing as neither the dependent or independent variables are known. As pointed out by Greene (2008, p. 868), if the interest is only the subpopulation of observed results, WLS (or REML) is appropriate after corrections for heteroskedasticity and interdependence. However, if inferences are to be extended beyond the subpopulation, WLS estimates are biased toward zero and a maximum likelihood procedure is more appropriate. Further, Figure 1 provides some information on where the data are missing or unobserved. In Table 4, columns (7)–(9) show bivariate and multivariate results from a truncated regression model, with the lower limit set at the minimum

observed value of the z -statistic. All of the intercepts are significantly positive and the precision slopes are insignificant. In column (9), the slope coefficient for promotion portrayal is significantly positive, but the dummy variable for limited number of advertising covariates also is significantly positive and large in magnitude. Because many of the promotion studies estimate models with a restricted number of variables, this result raises an important question regarding the accuracy of studies of ABMs and movies. Finally, the SSCI impact factor is significantly negative. Holding precision constant, this indicates that longitudinal studies published in lower-ranked journals contain larger values. The implication is that these journals tend to have more lax standards when it comes to model specification or statistical methods. Overall, the truncated model appears to improve on the WLS and REML estimates. The results indicate substantial selection bias associated with misspecification of the advertising–marketing covariates and with selection based on journal quality.

4. Publication Bias in Drinking Behaviour Studies

This section analyses publication bias for 40 estimates of the effects of alcohol marketing on drinking behaviour by adolescents. The analysis parallels the procedures used for drinking onset. Figure 2 shows the log odds ratios plotted against the standard errors (upper panel) and the inverse of the standard error or precision (lower panel). The filled funnel plots show the actual observations from Table 3 and imputed values obtained using the trim-and-fill procedure. There are eight imputed observations (filled dots) and the recomputed fixed-effect mean (filled diamond) is only 1.088 (95% CI, 1.060–1.116). The recomputed random-effects mean is 1.097 (1.040–1.156). The imputed values are concentrated in the lower-left portion of the diagrams, which again is consistent with publication bias in the positive direction. The Q and I^2 tests reject homogeneity ($Q = 110.5$, $p < 0.001$; $I^2 = 64.7$, $p < 0.001$).

4.1 WLS and REML Regression Models

The results for FAT–PET bivariate tests for publication bias are shown in Table 5. In columns (1) and (4), the bivariate intercept terms are significant and the precision coefficients are insignificant. There are four multivariate regressions estimated by WLS and REML. The M variables are represented by four weighted covariates for mass media specifications, promotion specifications, beer drinking as an outcome and binge drinking as an outcome. The K variables are represented by a binary variable for studies that include two or fewer advertising covariates, a binary variable for studies that do not include covariates for personality traits, and the SSCI impact factor for journal quality. The study by Stacy *et al.* (2004) accounts for 12 of the 40 observations but failed to include personality traits as a covariate, so the estimates are suspect due to omitted variable bias. Columns (2) and (3) show the results for WLS with cluster robust standard errors. The intercepts are significant in both regressions. In column (3), the positive effect for the ads covariate

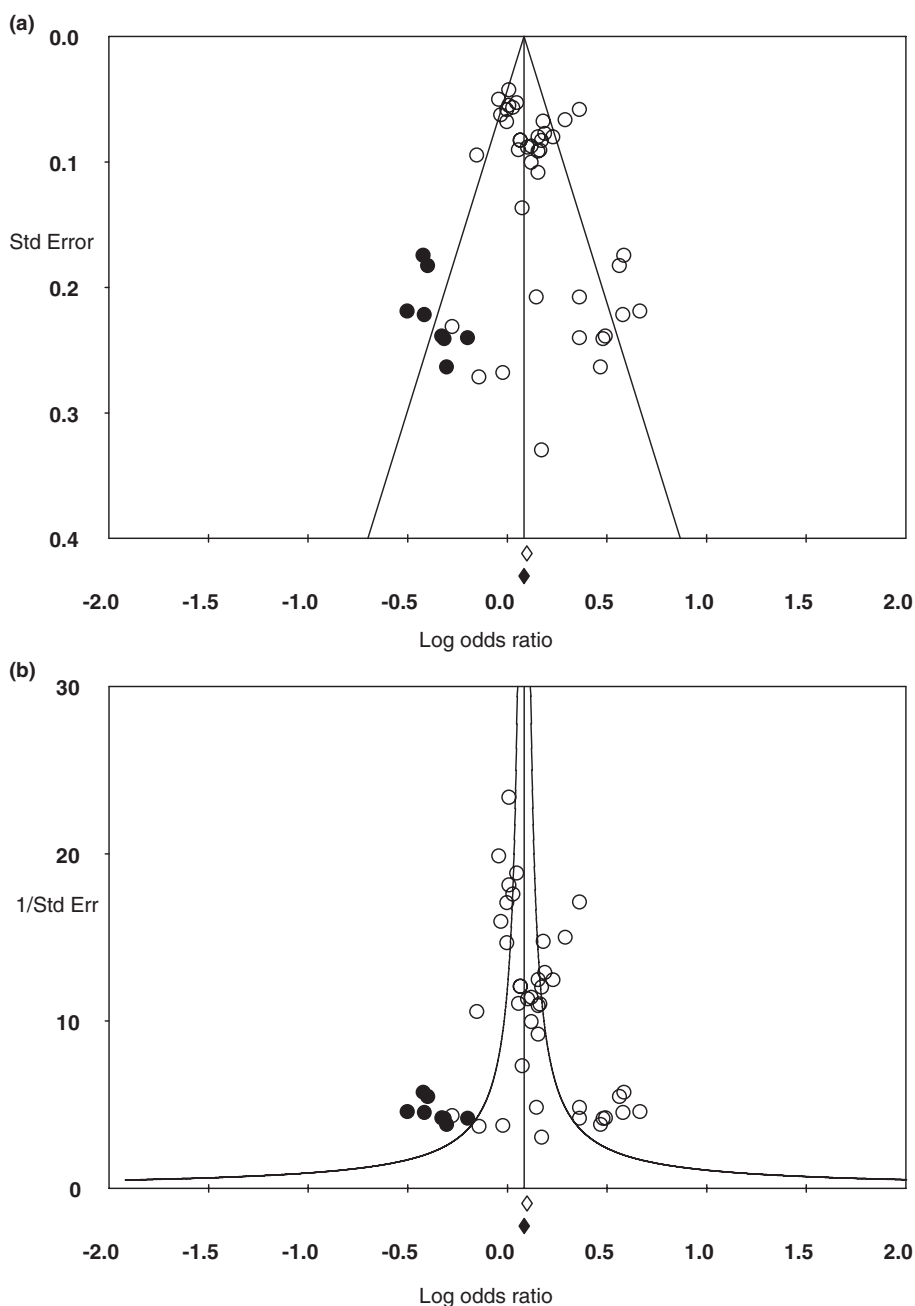


Figure 2. (a) Funnel Plot of Standard Error, Drinking Behaviours. (Filled dots are imputed studies; diamonds are means.) (b) Funnel Plot of Precision, Drinking Behaviours. (Filled dots are imputed studies; diamonds are means.)

Table 5. Meta-regressions for Drinking Behaviours and Publication Bias.

| Variables | (1) WLS | (2) WLS | (3) WLS | (4) REML | (5) REML | (6) REML | (7) Truncate | (8) Truncate | (9) Truncate |
|---------------------------|------------------------|------------------------|------------------------|------------------------|------------------------|------------------------|------------------------|------------------------|------------------------|
| Intercept | 1.533 (3.10) | 1.538 (3.72) | 2.987 (4.73) | 1.502 (2.61) | 1.686 (2.27) | 2.858 (2.44) | 1.737 (2.92) | 1.859 (5.13) | 3.046 (6.59) |
| Precision | -0.018 (0.26) | -0.061 (1.61) | -0.082 (1.36) | -0.021 (0.43) | -0.099 (1.42) | -0.082 (0.86) | -0.022 (0.32) | -0.107 (2.98) | -0.130 (2.11) |
| Mass media wt. | | 0.043 (1.25) | 0.046 (1.28) | | 0.047 (1.09) | 0.047 (1.07) | | 0.080 (3.57) | 0.082 (3.40) |
| Promotion portrayals wt. | | 0.032 (0.77) | 0.115 (1.39) | | 0.081 (1.18) | 0.113 (1.56) | | 0.065 (2.05) | 0.140 (2.37) |
| Beer drinking wt. | | 0.032 (0.59) | 0.049 (0.84) | | 0.073 (1.34) | 0.060 (1.00) | | 0.058 (1.22) | 0.079 (1.76) |
| Binge drinking wt. | | 0.025 (0.79) | -0.054 (2.33) | | -0.022 (0.29) | -0.051 (0.65) | | 0.023 (0.68) | -0.043 (1.69) |
| Personality traits binary | | | 1.049 (1.75) | | | 1.012 (1.23) | | | 0.963 (1.85) |
| Ads covariate binary | | | 1.443 (2.45) | | | 1.437 (1.28) | | | 1.335 (2.47) |
| SSCI impact factor | | | -0.687 (5.08) | | | -0.651 (1.71) | | | -0.582 (2.85) |
| Std errors | cluster | cluster | cluster | multi | multi | multi | cluster | cluster | cluster |
| R ² est. | 0.004 | 0.045 | 0.274 | — | — | — | — | — | — |
| N | 40 | 40 | 40 | 40 | 40 | 40 | 40 | 40 | 40 |

Notes: *T*- and *z*-statistics (absolute values) in parentheses; bold entries indicate significant coefficients at the 95% level. WLS regressions with cluster robust standard errors based on study id. REML are multi-level mixed-effects regressions, with standard errors based on restricted maximum likelihood estimation. Maximum-likelihood truncated regressions are restricted to minimum effect size or larger, with cluster robust standard error based on study id. All estimates obtained using Stata/IC 10.1.

Variables: Dependent variable is the *z*-statistic for the log odds ratio; Precision is the inverse of the standard error; Mass media is a binary variable for mass media exposure, weighted by the inverse standard error; Promotion portrayals is a wt. binary variable for branded merchandise and movie exposure; Beer drinking is a wt. binary variable for beer drinking as the outcome variable; Binge drinking is a wt. binary variable for binge drinking as the outcome variable; Personality traits is a binary variable if the study omits personality covariates; Ads covariate is a binary variable if the study has one or two variables for advertising and marketing exposure; and SSCI impact factor is the ISI-JCR index of journal quality (see Murtaugh, 2002).

is consistent with specification bias and the negative coefficient for the SSCI impact factor again indicates selection bias associated with journal quality. REML regressions yield significant intercepts and insignificant precision slopes in all three cases.

4.2 *Truncated Regression Model for Drinking Behaviours*

The truncated regressions provide a slightly different picture. In column (7), the intercept is significant and the precision slope is insignificant, which replicates the results for WLS and REML models. However, in columns (8) and (9), the precision slopes are significantly negative. In regression (9), the dummy variable for the number of advertising covariates is significantly positive, and its magnitude is substantial. The SSCI variable for journal quality is significantly negative, indicating that lower-ranked journals contain larger primary effect sizes, other things being equal. The personality trait variable is positive, but insignificant. Overall, the results in Table 5 are consistent with publication bias in the positive direction, omitted variable bias in some studies, and publication selection by lower-ranked journals. Genuine effects in the MRA model are given by combinations of the M variables and the precision term (Doucouliagos and Stanley, 2009). When all K variables are set equal to zero in regression (9), the net effect of mass media is negative ($-0.130 + 0.082 = -0.048$) and the net effect of promotion portrayals is a small positive value ($-0.130 + 0.140 = 0.010$). Comparing these values to the positive effects due to selection bias and the intercept term indicates that primary effect sizes for drinking behaviours are dominated by publication biases.

4.3 *Summary of Results for Publication Bias*

In summary, there are five results in the present study that are indicative of publication bias in the literature on alcohol marketing and adolescent drinking. First, some published studies contain specification errors due to omitted variables, which are ignored by researchers and reviewers. This is especially the case for the primary studies that examine the effects of ABMs and cinema portrayals of alcohol. Second, random-effect and unweighted means are larger in magnitude compared to fixed-effect means. This implies that more precise studies have effect sizes that are smaller in magnitude. Third, funnel plots of the effect sizes are asymmetric and the filled values are concentrated in the lower left-hand portion of the diagrams, which means that less precise estimates tend to account for the asymmetry. Fourth, six bivariate regressions yield significant intercepts and insignificant precision slopes for both drinking onset and drinking behaviours. The FAT–PET tests indicate bias in every instance. Fifth, the multivariate regressions account for both study-level heterogeneity and publication selection bias. Heterogeneity is represented by weighted explanatory variables for mass media, promotion portrayals, beer drinking and binge drinking. Publication selection is represented by unweighted explanatory variables for restricted advertising–marketing specifications, omitted

personality traits and the SSCI index of journal quality. In virtually all cases, the intercept values are significantly positive. The results for the precision slope are insignificant in 10 of 12 cases, and significantly negative in two cases. The insignificant results are consistent with lack of genuine effects of marketing on adolescent drinking, while the significant negative results are used to demonstrate that studies of drinking behaviours are dominated by selection biases for both mass media and promotion portrayals.

5. Dissemination Bias in Adolescent Drinking Studies

Although many aspects of dissemination bias have been distinguished, one of the more difficult issues is the suppression of data or misuse of results due to competing interests and agendas (Young *et al.*, 2008). For instance, Halpern and Berlin (2005, p. 313) argue 'if data suppression due to competing interests were influencing the evidence available for inclusion in a meta-analysis, then simple inspection should reveal that the funnel plot among studies funded by for-profit organizations has greater asymmetry than the plots of either unfunded studies or those funded by non-profit organizations'. Although a funding issue might be relevant for alcohol research, it was not possible to investigate its impact in the present study.¹³ Instead, a related issue is discussed, which is selection bias in the interpretation and use of results by researchers and health policy interest groups. Rothstein *et al.* (2005) refers to this influence as 'outcome reporting bias', while Song *et al.* (2000) suggest the term 'dissemination bias' to refer generally to the use of empirical results depending on the direction and strength of research findings. This section provides examples of dissemination bias using longitudinal studies, cross-section studies and evaluative statements in the alcohol policy literature. No claim is made that the survey is comprehensive. There is evidence of bias, however, because empirical results can be shown to contradict what is reported in both the research and policy literatures on adolescent drinking.

Table 6 contains a summary of results from 15 primary studies of adolescent drinking and advertising, including 10 longitudinal studies and five cross-sectional or panel data studies. In each case, a summary of results is provided together with evaluative statements by the investigators or outside users of the research results, such as health policy interest groups. Several evaluative statements are provided by the advocacy organization, Center on Alcohol Marketing and Youth (CAMY) (2006, 2007a, b). A number of basic inconsistencies are demonstrated by the table results. Most of the primary studies contain insignificant or significantly negative results (e.g. Austin *et al.*, 2006, for magazines; Fleming *et al.*, 2004, for billboards), but these results are usually ignored by investigators and users. Several studies contain specification errors, but these omissions are universally ignored by users. Ignoring null results or misspecification problems is evidence of dissemination bias by investigators and interest groups. As demonstrated above, misspecified models tend to yield larger effect-size estimates, while null results are common in the primary studies.

Table 6. Narrative Analysis of Dissemination Bias in Adolescent Drinking Studies.

| Study and data | Summary of selected results | Conflicting evaluative statements |
|--|---|--|
| Connolly <i>et al.</i> (1994) Longitudinal, New Zealand, Youth, ages 13, 15 and 18 | Study reports 48 estimates of the effects of ad recall on drinking amt, frequency and max amt for males and females at age 18. Only 3 of 48 estimates are significantly positive ($p < 0.05$). For TV viewing, only 3 of 12 estimates are significant | Study concludes that the results are 'important for policy makers' (p. 1262). Paper is cited in Casswell (2004, p. 473) as an example of a study supporting a 'causal impact of advertising'. Also cited in Martin <i>et al.</i> (2002, p. 905) as supporting a causal link. Also cited in Nunez-Smith <i>et al.</i> (2008) as demonstrating TV viewing predicts use |
| Grube and Wallack (1994) Cross-sectional, California, Youth, ages 10–14 | Following a specification search, several ad variables are dropped because they are 'unrelated to any of the endogenous variables' (p. 256) measuring drinking intentions as an adult | The dropped variables include hours of weekend afternoon TV viewing and hours of weekday TV viewing. Study is cited by Hastings <i>et al.</i> (2005, p. 302) as showing beer ads linked to 'intentions to drink as an adult', but the omitted variables are ignored |
| Robinson <i>et al.</i> (1998) Longitudinal, San Jose, CA, Youth, ages 14–15 and 16–17 | Null results for 2 of 4 media for drinking onset (VCR videos, computer games). Null results for drinking maintenance for all 4 media (TV, music videos, VCR videos, computer games) | Study concludes 'there were no significant associations between baseline media use and maintenance of drinking' (p. 54). Paper is cited by Strasburger (2002, p. 367) as supporting 'certain media use as a possible cause of early alcohol use', but all null results ignored |
| Gentile <i>et al.</i> (2001) Cross-sectional, Midwest USA, Youth, ages 12–19 | Study omits advertising variables for hours of TV watched and number of sports programmes watched for frequency of drinking. Coefficient estimates and p -values are unreported for all covariates in the study. No market variables for five dispersed areas | Paper is cited by AlcoholPolicyMD.com (2005) as demonstrating that 'media and advertisements are perhaps the most significant predictors of adolescents'... current drinking behaviour. The identical claim appears in CSPI (2005, p. 10), but no advertising variables are reported in Gentile <i>et al.</i> for frequency of drinking |

- Fleming, Thorson and Atkin (2004), Cross-sectional, USA nationwide telephone survey, Youth, ages 15–20, and young adults, ages 21–29
- Liquor ads on billboards and in magazines are unrelated to positive alcohol expectancies, youths' intentions to drink and alcohol use by young adults. TV beer ads have a significant *negative* association with youths' alcohol expectancies and radio ads for liquor have a *negative* association with drinking intentions
- Stacy *et al.* (2004) Longitudinal, Los Angeles, Youth, ages 12–13 and 13–14
- Null results for cued-recall memory test for all three drinking outcomes (beer, wine/liquor, 3-drink episodes). Draw-an-event memory test is insignificant for wine/liquor use and 3-drink episodes, and *negative* for beer use. Overall, only 6 of 12 advertising covariates are significant
- Ellickson *et al.* (2005) Longitudinal, South Dakota, Youth, ages 12–13 and 14–15
- Null results for the TV beer ads and magazine alcohol ads for drinking onset. Null results for TV beer ads and in-store ads for drinking frequency. Significantly *negative* effect of TV viewing on both drinking onset and frequency. Some results conflict with Collins *et al.* (2007)
- Study concludes that 'greater exposure to alcohol advertising... was not a determining factor that predicted the 15–20 year olds' intentions to drink and young people's... consumption' (p. 23). However, the abstract to the paper reports that 'alcohol advertising was influential in shaping young people's attitudes and perceptions about alcohol advertising messages' (p. 3). Cited by CAMY (2007b) as showing that alcohol ads shape attitudes and positive expectancies for youth
- Study concludes that 'the weight of the evidence from this study is consistent with that of some other studies suggesting that exposure to alcohol advertising increases the risk of subsequent alcohol use... even if the risk attributable to advertising is small relative to other influences' (p. 507). Study is cited by Martin *et al.* (2002, p. 905) as illustrating 'innovative approaches to measuring the recall of alcohol advertising that may overcome some... problems'
- Study concludes there is 'no evidence that exposure to television beer advertising affects subsequent drinking... [but] results are consistent with a process in which adolescents start to drink in response to advertising' (p. 244). Paper is cited by CAMY (2006) in a TV report as showing that 'the more alcohol advertising young people are exposed to, the more likely they are to drink or drink more' (p. 3)

Table 6. *Continued.*

| Study and data | Summary of selected results | Conflicting evaluative statements |
|---|--|--|
| Austin, Chen and Grube (2006), Cross-sectional, San Francisco, Youth, ages 9–17 | Reading magazines has a significant positive effect on scepticism about alcohol ads, which has a positive effect on <i>negative</i> alcohol expectancies. Negative expectancies in the path analysis model have a significant <i>negative</i> impact on alcohol use for males and females, and for all youth ages 12–17 years | Study concludes that ‘youth who read magazines more often were more sceptical about advertising’ (p. 381), and ‘scepticism may indirectly prevent underage drinking’ (p. 382). Paper is cited by Siegel <i>et al.</i> (2008) as suggesting that for magazines ‘exposure to alcohol advertising may affect [adversely] the drinking-related attitudes, intentions and behaviour of adolescents’ (p. 484). |
| McClure <i>et al.</i> (2006), Sargent <i>et al.</i> (2006) Longitudinal, NH and VT, Youth, ages 10–14 | Identical samples are used in the two studies. In McClure <i>et al.</i> , ownership of ABMs (at follow-up) is positively related to drinking onset at follow-up. In Sargent <i>et al.</i> , baseline no. of hours of exposure to alcohol portrayals in movies is a significant predictor of drinking onset and prevalence of use by drinkers | Neither study mentions the possible use of both ABMs and movie-use in the same regression or considers the possible biases due to omission of other forms of advertising and marketing. The possibility of omitted variables bias in this and other studies is overlooked in reports by CAMY (2007b) and Ostroff and Jernigan (2007) |
| Saffer and Dave (2006), Panel data, MTF and NLSY, Youth, ages 12–17 and 12–16 | Local advertising expenditures is insignificant for three MTF drinking measures for blacks (past year, past month, bingeing) and in two cases for males (past month, bingeing). In NLSY regressions, log ads is insignificant for past month participation | The study concludes that a ‘reduction of alcohol advertising can produce a modest decline in adolescent alcohol consumption, though effects may vary by race and gender’ (p. 617). The authors’ cautionary statement is ignored by CAMY (2007b) and Jernigan (2009, p. 10) |

| | | |
|---|---|--|
| Collins <i>et al.</i> (2007) Longitudinal, South Dakota, Youth, ages 11–12 and 12–13 | Null results for beer drinking for ESPN beer ads, magazine reading, radio listening, concessions, in-store displays, weekly TV viewing and other TV ads. For drinking intentions, only two of nine advertising–marketing covariates are significantly positive. Some results conflict with Ellickson <i>et al.</i> (2005) | Study concludes that ‘individual effect sizes for most forms of advertising were small, and some types of advertising appear to have no effect’ (p. 533). Cited in Jernigan (2009, p. 10) as showing that ‘television beer advertisements [and] alcohol advertisements in magazines . . . were strongly predictive of drinking and intentions to drink’ |
| Pasch <i>et al.</i> (2007) Longitudinal, Chicago, Youth, ages 11–12 and 13–14 | Null results for outdoor alcohol ads for alcohol behaviour for all students, grade-6 non-drinkers and grade-6 drinkers (nine regressions for different outdoor ad measures) | Study concludes that ‘we did not find a statistically significant association between exposure to outdoor alcohol advertising and the alcohol behaviours subscale’ (p. 594). Paper is cited in Ostroff and Jernigan (2007, p. 1) as showing that ‘outdoor signage . . . predicted likelihood of subsequent drinking’. The identical claim appears in the CAMY (2007) report on TV and magazine advertising |
| McClure <i>et al.</i> (2009), Wills <i>et al.</i> (2009), Longitudinal, Nationwide USA, Youth, ages 10–14 and 12–16 | Exposure to alcohol portrayals in movies and viewing of TV are unreported covariates in the study of ABMs by McClure <i>et al.</i> (2009). None of these variables is used in the study of movie portrayals by Wills <i>et al.</i> (2009) using the same sample | Anderson <i>et al.</i> (2009, p. 14) conclude that ‘whilst all the studies to some extent, measured and controlled for other variables likely to be associated with drinking uptake, it is impossible to know [sic] if all relevant variables were measured and adjusted for’ (p. 14) |

Abbreviations: ABM is alcohol-branded merchandise; CAMY is Center on Alcohol Marketing and Youth; CSPI is Center on Science in the Public Interest; MTF is Monitoring the Future; and NLSY is National Longitudinal Survey of Youth.

5.1 *Four Examples of Dissemination Bias*

Four examples from the table illustrate the nature and extent of this problem. First, for New Zealand youth, Connolly *et al.* (1994) report 48 estimates of the effects of advertising exposure on drinking amounts, drinking frequency and maximum amount of drinking by males and females. Only three of the 48 estimates are statistically positive, which could easily occur by chance. The study also reports 12 estimates for the effect of TV viewing on drinking behaviours, and only three of these are significantly positive. Two recall estimates for females are significantly *negative*. However, the results are cited by Casswell (2004) as an example of a study that supports a causal link between advertising and drinking. Specifically, Casswell (2004, p. 473) states that ‘supporting evidence has been found in a series of longitudinal analyses of data from a cohort of New Zealand teenagers’. No mention of null (or negative) results in the New Zealand studies is revealed by Casswell’s evaluation of these studies, in which she also was an investigator. This is a clear example of dissemination bias.

A second example is provided by a cross-sectional study by Fleming *et al.* (2004), which uses a nationwide telephone survey of youth (ages 15–20) and young adults (ages 21–29) to examine the effects of exposure to four mass media (TV, radio, magazines, billboards) and the influence of attitudes and perceptions about liquor ads. The chain of causality for the perception variables is unclear, and the study does not attend to possible endogeneity or reverse causation associated with these variables (Heckman *et al.*, 2008). A particular specification problem in this study is the omission of market-wide variables for alcohol prices and regulations. For positive alcohol expectancies, none of the mass media variables are significant, and the variable for TV ads for beer is significantly *negative* for youth. When the perception variables are added to the regression, the results for the mass media variables are unchanged. In this study, any possible effects of advertising are indirect, but the magnitude and significance of the mediated indirect effects are not reported (Nelson, 2001). The study concludes that ‘greater exposure to alcohol advertising... was not the determining factor that predicted the 15–20 year olds’ intentions to drink and the young adults’ consumption’ (Fleming *et al.*, 2004, p. 23). This study is cited by CAMY (2007b) as showing that alcohol ads shape attitudes and expectancies, but this ignores the fact that all direct effects of the ads are insignificant. For the attitude and perception variables, Fleming *et al.* (2004, p. 15) report 30 estimates of the indirect effects of advertising: six coefficients are significantly positive; two coefficients are significantly *negative* (radio, billboards); and 22 are insignificant. This is a very weak basis for claiming that this study provides evidence of a positive effect of advertising messages on attitudes, perceptions or expectancies.

The third example is the longitudinal study by Ellickson *et al.* (2005), which uses a sample of South Dakota youth. Results are reported for drinking onset and frequency. Null results are reported for TV beer ads and magazine ads for drinking onset. Null results are reported for TV beer ads and in-store ads for drinking frequency. The effect of TV viewing is significantly *negative* for both

drinking onset and frequency at grade 9. The main positive result in the study is the relationship for in-store displays and drinking onset. However, only two of five advertising coefficients for drinking frequency are significantly positive; small positive coefficients are reported for magazine ads and concession stands. The authors report that their study found 'no evidence that exposure to television beer advertising affects subsequent drinking' (Ellickson *et al.*, 2005, p. 244). Despite this conclusion, the study is cited by CAMY (2006, p. 3) in its report on TV exposure as showing that 'the more alcohol advertising young people are exposed to, the more likely they are to drink or drink more'. Null and negative results in this and other studies are totally ignored in the CAMY reports.

The fourth example is provided by the longitudinal study by Collins *et al.* (2007), which also uses the sample of South Dakota youth. The differences between the two studies are (1) different age groups (grade 7 drinking rather than grade 9); (2) different statistical models for drinking frequency; and (3) different sets of covariates (e.g. TV shows in Ellickson *et al.*, 2005; TV sports ads in Collins *et al.*, 2007). For past-year drinking (any amount), the null results in Collins *et al.* (2007) include ESPN-TV beer ads, other TV beer ads, weekly TV viewing, magazine reading, radio listening, beer concessions, and in-store displays. The null results for in-store displays conflict with results in Ellickson *et al.* (2005). For drinking intentions, the null results include ESPN-TV beers ads, other sports beer ads, weekly TV viewing, magazine reading, radio listening, beer concessions and in-store beer displays. Combining the results, 18 coefficients were estimated for advertising and marketing of alcohol, and only four are significantly positive.¹⁴ The study concludes that 'individual effect sizes for most forms of advertising were small, and some types of advertising appear to have no effect' (Collins *et al.*, 2007, p. 533). Despite this conclusion, the study is cited by Jernigan (2009, p. 10) as showing that 'television beer advertisements [and] alcohol advertisements in magazines . . . was strongly predictive of drinking and intentions to drink'. This is another clear example of overreaching in the health policy literature on adolescent drinking.

The remaining 11 studies in Table 6 and the other evaluative statements are consistent with this brief review. Similar problems exist for reports by government agencies, such as the reports of the Federal Trade Commission (FTC) to the US Congress on alcohol marketing and advertising (1999, 2003, 2008). The more recent FTC studies (2003, 2008) corrected some of the problems associated with CAMY's method for determining advertising exposure, but the scientific basis for the FTC reports is incomplete and misleading. None of the reports contains a thorough or up-to-date literature review, and the first FTC report (1999, p. 4) merely stated that for underage drinking decisions, 'there is reason to believe that advertising plays a role'. It is difficult to see how good public policy can be based on non-transparent claims or misleading citations of scientific papers.¹⁵ In summary, dissemination bias is a serious problem in the literature on adolescent drinking and marketing of alcohol, which should be addressed in future studies, literature reviews and funding priorities.

6. Conclusions

As noted in prior reviews, the effect of alcohol marketing on adolescent drinking is modest, but the evidence indicates that it may not exist at all for mass media and other exposures. A meta-analysis reveals three problems in the existing literature. First, empirical results in the primary studies are mixed and inconclusive. Some studies find significant results for one or two covariates that measure marketing exposure, but the same variables are insignificant or negative in other studies (e.g. Robinson *et al.*, 1998; Ellickson *et al.*, 2005, for TV viewing). Some studies find significant results for a particular age/gender cohort, but other studies provide conflicting results (Stacy *et al.*, 2004; Saffer and Dave, 2006, for binge drinking by teenagers). Even studies using identical data can yield conflicting result (Ellickson *et al.*, 2005; Collins *et al.*, 2007, for in-store displays). Only 21 of 63 estimates (33%) are statistically significant. Second, an examination of comparable results from logistic studies reveals evidence that is consistent with publication bias and misspecification of empirical models. Filled funnel plots indicate that reported results are biased in the positive direction, which implies that weighted means are too large in magnitude. Bivariate tests provide empirical evidence that is consistent with publication bias and absence of genuine effects. The MRA multivariate model for publication and specification bias also indicates that these problems exist in the primary studies. Publication bias implies that the sample is truncated, with the lower limit on the observations revealed by the funnel plots. WLS estimates in the presence of truncation are biased toward zero since the model is misspecified. Truncated regression models estimated by restricted maximum likelihood also demonstrate the deleterious effects of model specification and journal quality. All of the MRA models demonstrate the importance of publication bias for both drinking onset and drinking behaviours, and cast doubt on any causal interpretation of the primary results. Third, a narrative review of youth drinking studies shows that dissemination bias exists in the public health policy literature. This is especially true for the reports issued by CAMY, but the problem is widespread. What can be done in light of these problems? It would be beneficial for empirical studies to adopt better standards for model specification and reporting of results, such as the inclusion of market-wide variables. Studies that cover only one or two marketing methods are incomplete on specification grounds. Studies should report full results for covariates, preliminary regressions and sensitivity analyses. A greater degree of replication should exist across future studies. This is especially true for the advertising and marketing covariates, where there is substantial diversity in the variables that measure exposure to commercial messages and images. Multicausal models need to be developed that treat marketing exposure as an endogenous variable (Geweke and Martin, 2002; Heckman *et al.*, 2008; Nelson, 2010b). A great deal of work remains to be done if this literature is to serve as a basis for sound public policy. These problems are not apparent in the recent reviews by Anderson *et al.* (2009), Gordon *et al.* (2009) and Smith and Foxcroft (2009) or, for that matter, in the earlier surveys by the National Institute on Alcohol Abuse and Alcoholism (2000) and the National Research Council (2004).

A problem encountered in the present study was the inability to explain a large portion of the variation in z -statistics for either drinking onset or drinking behaviours. There are several possible explanations for this outcome. First, the samples are small and there is diversity in model specifications for advertising and marketing covariates. This suggests that the degree of replication necessary for the MRA model may be lacking, but the situation is improved somewhat by using a truncated regression model. Second, the statistical model used for z -statistics captures biases that exist in the estimation of regression coefficients and standard errors. However, this problem also exists for standard errors, such as omission of robust errors or failure to correct for spatial correlation (Stanley, 2008). Third, it might be beneficial to focus on the more precise studies – those in the upper portion of the funnel plots – which can provide a better measure of the quality of the estimates or at least serve as a complement to the usual tests of statistical significance that are emphasized in economics (Stanley and Doucouliagos, 2009). Fourth, it may be that each empirical study should be viewed as a unique case study. The influences on adolescent alcohol behaviours in California might be quite different from other parts of the USA or worldwide. In this case, policy generalizations are difficult or impossible. Future primary research might help resolve this issue. Last, it is important to keep in mind that advertising regulation is one of several possible policy tools to combat underage drinking. The report by Babor *et al.* (2003), sponsored by the World Health Organization, concluded that advertising bans and other marketing regulations were among the least effective policy strategies. The report also notes that ‘the knowledge needed to address health and social problems is unlikely to reside in a single discipline or research methodology’ (Babor *et al.*, 2003, p. 272). The present study adds support for both of these conclusions.

Notes

1. Longitudinal studies of adolescent alcohol behaviours are part of a broad literature that examines the possible influence of marketing exposure on youth alcohol beliefs, susceptibility, expectancies, intentions and actual drinking outcomes. There are a large number of narrative reviews of this literature, including Anderson (2007), Babor *et al.* (2003), Gordon *et al.* (2009), Grube (2004), Hastings *et al.* (2005), Martin *et al.* (2002) and Strasburger (2002).
2. Sutton *et al.* (2000, p. 8) argue that systematic studies ‘help us see more clearly where there are [research] gaps...[and] are more cumulative and more critically robust’. For other analyses of publication bias in systematic reviews, see Egger *et al.* (1997), Song *et al.* (2000) and Ioannidis and Trikalinos (2007). For general discussions of meta-analysis and publication bias, see Borenstein *et al.* (2009) and Roberts and Stanley (2005).
3. For example, Card and Kruger (1995) attribute publication bias to three sources: (1) reviewers and journal editors may be predisposed to accept papers that support conventional views; (2) reviewers and journals tend to favour papers with statistically significant results and (3) researchers use t -statistics of two or more for the main covariates as a guide for model specification and selection (or p -values of 0.05 or

less). A fourth factor is that papers with less conventional results are likely to be held to a generally higher statistical or econometric standard by reviewers. These factors are investigated below by including a variable for 'journal quality' in the meta-regression analysis.

4. An odds ratio is a measure of relative risk given by the probability of an event occurring in one group divided by the probability of it not occurring in another group, such as differences in exposure to alcohol marketing and the onset of drinking. An odds ratio that is significantly greater than one implies that an event is more likely to happen in the first group. It is standard practice in the longitudinal literature to use a 95% CI, which also is adopted in the present paper. Some results reported below are sensitive to this assumption.
5. The logistic study by Ellickson *et al.* (2005) did not report standard errors. Phyllis Ellickson (personal correspondence) provided this information and also confirmed a typographical error in the reported results for two marketing variables, i.e. for grade 7 non-drinkers, the reported coefficients are reversed for beer concession stands and in-store displays (see Table 2 later for corrected estimates).
6. Odds ratios and relative risk ratios are related, but they are not identical. The odds ratio approaches the risk ratio asymptotically at low risk levels and the difference is very small at an absolute risk of 10% or smaller. Some researchers suggest that below a threshold of 20%–30% the difference between odds ratios and relative risk ratios is unlikely to be important (Prasad *et al.*, 2008). This threshold fits the present sample.
7. Ellickson *et al.* (2005) also report results for several non-advertising variables for TV shows that are designed to capture adolescents' desire to be 'more mature' (i.e. viewing of MTV, Jerry Springer and Loveline). According to Ellickson *et al.* (2005, p. 239), 'none of these [TV shows] aired beer or other alcohol advertising during the relevant period'. These estimates are omitted because they are not comparable and better classified as 'personality traits'.
8. Using the unweighted values as a guide, odds ratios less than 1.10 are small; 1.10 to 1.30 is modest; 1.30 to 1.60 is large; and odds ratios greater than 1.60 are substantial.
9. The diagonal lines in Figure 1(a) and the curved lines in Figure 1(b) are 95% CIs for each standard error or precision estimate on the vertical axis. The width of the lines is an indication of heteroskedasticity. Points that lie to the left (right) of the lines indicate odds ratios that are smaller (larger) than expected under the fixed-effect assumption (Borenstein, 2005; Sterne and Egger, 2005). Funnel plots and the trim-and-fill procedure has been the subject of recent criticism as definitive methods. Regression-based procedures such as those employed below are recommended in Moreno *et al.* (2009a, b).
10. An alternative test is a regression of the z-statistic on the sample size or degrees of freedom as proposed in Card and Kruger (1995) and Macaskill *et al.* (2001). This test has lower power than Egger's test (Sterne and Egger, 2005).
11. Doucouliagos and Stanley (2008) argue that FAT-intercept values greater than 2.0 are indicative of severe bias, while values between 1.0 and 2.0 indicate substantial bias. For drinking onset and behaviour, the multivariate intercept values in the present study are close to or greater than 2.0.
12. For example, all of the primary studies include at least 13 covariates, except one study. All of the studies cover youth in the age range 14–16 years. All studies cover small geographic areas, except two. As a sensitivity analysis, I experimented with

- several other study-level regressors, including year of publication, year of sampling, and a non-US dummy. None of these variables were consistently significant or were subject to interpretation problems due to collinearity with the reported regressors.
13. All 12 studies included in the meta-analysis received funding from government agencies or non-profit groups, e.g. National Institute of Alcohol Abuse and Alcoholism, National Cancer Institute, National Institutes of Health, FRG Ministry of Health etc.
 14. Combining the results in Ellickson *et al.* (2005) and Collins *et al.* (2007), there are 14 marketing estimates for drinking onset and intentions, which are significantly positive in three cases, *negative* in one case (weekly TV viewing), and insignificant in 10 cases. For drinking frequency and past-year beer drinking, there are 14 marketing estimates, which are significantly positive in four cases, *negative* in one case (weekly TV viewing) and insignificant in nine cases. Overall, there are 28 estimates of marketing exposure and youth drinking, which are insignificant or negative in 21 cases (75%).
 15. A recent EC Health Forum report summarizes the two systematic studies, but also omits most null results (European Alcohol and Health Forum, 2009). The report was accompanied by a call for an advertising ban in the mass media and sports sponsorships (Anderson, 2009). See Nelson (2008, 2010a) and Paschall *et al.* (2009) for recent empirical evidence indicating null effects of advertising bans and sports sponsorship in the mass media for alcohol consumption by adults and youth. Another basic problem in longitudinal studies is the lack of a defined or measured relationship between individual marketing exposure and actual advertising expenditures or regulatory policies. For example, it is impossible to tell how much youth drinking would be affected by a ban on sports sponsorships (see Nelson, 2010b).

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